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DIAGNOSTIC TECHNIQUES

Observations on the Antidromic Type of Circus Movement Tachycardia in the Wolff-Parkinson-White Syndrome

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In the differential diagnosis of tachycardias showing a wide QRS complex and having a 1 to 1 relation between ventricular and atrial events, a supraventricular tachycardia with anterograde conduction over an accessory pathway and retrograde conduction by way of the specific conduction system must be considered. Five patients showing this type of circus movement tachycardia were studied by programmed electrical stimulation of the heart. Sudden changes in the tachycardia cycle length were observed in these patients that were based on changes in the VH interval. This finding suggested a change in the reentrant circuit with anterograde conduction over the accessory pathway but retrograde conduction sometimes occurring over the right bundle branch and at other times over one of the two divisions of the left bundle branch system. Characteristically, the tachycardia cycle length changed suddenly depending on the bundle branch used in retrograde direction.

In one patient, an important difference was also observed between the anterograde effective refractory period of the accessory bypass (280 ms) and the shortest RR interval between preexcited QRS complexes during atrial fibrillation (measuring 190 ms). It is postulated that the short RR intervals during atrial fibrillation in the Wolff-Parkinson-White syndrome could result from bundle branch reentry after activation of the ventricles over the accessory pathway.

The most common tachycardia in patients with an accessory atrioventricular (AV) pathway is a circus movement tachycardia using the normal AV conduction system as the anterograde limb and the bypass tract as the retrograde limb of the reentrant circuit. This is the so-called orthodromic circus movement tachycardia (1,2). Atrial fibrillation and flutter are less common, but they are potentially more life-threatening because extremely rapid ventricular rates may result and precipitate ventricular fibrillation in the presence of a short refractory period of the accessory pathway (3,4).

A circus movement tachycardia in patients with the Wolff-Parkinson-White syndrome, using in anterograde manner the accessory bypass and in retrograde manner the AV node, is less common than the orthodromic type. Recognition of the antidromic type of tachycardia, however, is of clinical interest because it can be extremely difficult to differentiate electrocardiographically this type of tachycardia from ventricular tachycardia (5).

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Five patients with the Wolff-Parkinson-White syndrome and the antidromic type of circus movement are presented in this report. Tachycardia rate changed suddenly in relation to changes in ventriculoatrial (VA) conduction time during the arrhythmia. This change in VA conduction time was based on the use of different parts of the bundle branch system as the retrograde limb of the reentrant circuit. In one of these patients bundle branch reentrant beats were considered responsible for the unexpected short cycles observed at the ventricular level during atrial fibrillation.

Methods

Patients. This study group consisted of five patients with the Wolff-Parkinson-White syndrome, aged 18 to 54 years (mean 35). There were four men and one woman. After obtaining informed consent an electrophysiologic study was performed in the nonsedated state at rest. The patients were not receiving medication at the time of study.

Electrophysiologic study. Catheters were passed through both femoral veins using the Seldinger technique and positioned in the heart under fluoroscopic guidance. These included quadripolar catheters in the high right atrium and coronary sinus, a bipolar catheter in the right ventricular apex and a bipolar catheter placed across the tricuspid valve for recording of a His bundle electrogram. The methods for

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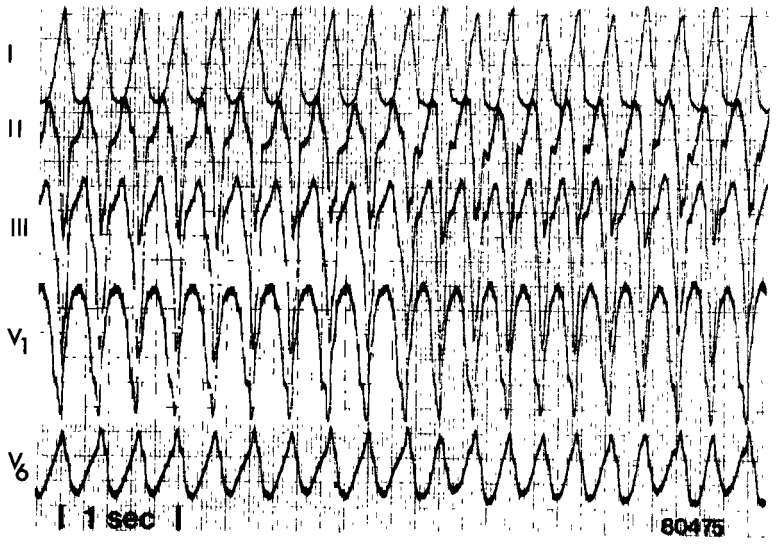


Figure 1. Case 1. Five surface lead electrocardiogram (I, II, III, V_1, V_6) during an episode of a tachycardia with a wide QRS complex. Note in the middle of lead II the sudden occurrence of a negative wedge in the ST-T segment, which is accompanied by an increased rate of the tachycardia.

stimulation and recording used in our laboratory and the definitions used in this report have previously been described in detail (6). All patients underwent a complete electrophysiologic investigation. Only the data relevant to this article will be presented.

Results

Features of antidromic tachycardia. The circus movement tachycardia, which could be initiated in all five patients by single premature beats from the high right atrium and the coronary sinus, used in an anterograde direction the accessory pathway and in a retrograde direction the normal AV conducting system (the antidromic type). Figure 1 shows a representative example of the surface electrocardiogram of Patient 1 with a right-sided accessory pathway during an episode of tachycardia with a wide QRS complex. During this tachycardia a sudden change in the ST-T segment suggesting a negative P wave, particularly in lead II, can be observed. This change in configuration of the ST-T segment is accompanied by an increase in rate of the tachycardia without a change in the QRS complex. The negative deflection corresponded with retrograde atrial activation as observed in the endocavitary leads and was not seen during the tachycardia with the slower rate. This phenomenon, which was present in all five patients, will be analyzed in detail in this patient.

The endocavitary recordings from Patient 1 (Fig. 2) show that the appearance of the negative deflection on the surface electrocardiogram and the decrease of tachycardia cycle length from 325 to 275 ms is accompanied by a shortening in the VA interval from 210 to 160 ms. As indicated in the His bundle recording this shortening in the VA interval is caused by a shortening in the VH interval from 160 ms during a tachycardia cycle length of 325 ms to 110 ms during a tachycardia cycle length of 275 ms. The HA interval remained constant. A single atrial premature beat delivered during tachycardia was able to advance ventricular activity

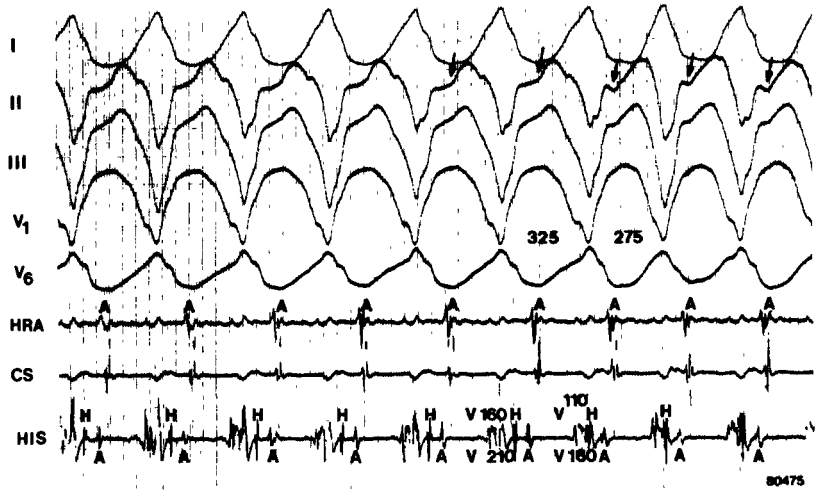


Figure 2. Case 1. The same tachycardia shown in Figure 1. Five surface electrocardiographic leads (I, II, III, V_1, V_6) are recorded simultaneously with three additional endocavitary leads from the high right atrium (HRA), coronary sinus (CS) and His bundle area (His). As indicated by the **arrows**, at the time of the sudden change of tachycardia cycle length from 325 to 275 ms, a negative P wave can be identified in lead II, which was obscured at the longer cycle length in the ST segment. As shown in the His bundle lead, this shortening of the tachycardia cycle by 50 ms is due to a shortening in the ventriculoatrial (VA) conduction time from 210 to 160 ms. This change in VA conduction time is caused by a decrease in the VH interval from 160 to 110 ms. In this and subsequent figures endocavitary electrograms have been retouched for clarity.

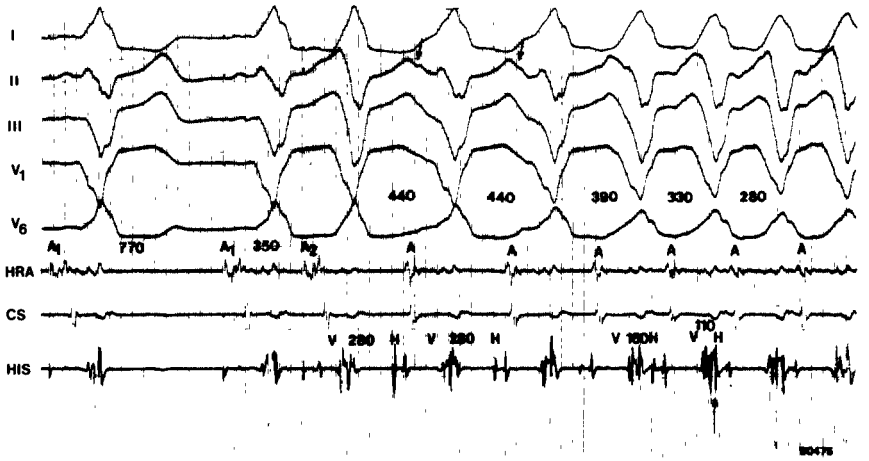


Figure 3. Case 1. Initiation of the antidromic type of circus movement tachycardia by an atrial premature beat after 350 ms during pacing from the right atrium with a basic cycle length of 770 ms. Note that the first two cycles of the tachycardia have a cycle length of 440 ms because of a very long VH interval of 280 ms. After these two cycles, there is a shortening of the tachycardia cycle length because of shortening of the VH interval. The **two small arrows** indicate the negative P wave in lead II during the long cycles. The **long arrow (below, right)** indicates the retrograde His bundle deflection, obscured in the ventricular complex during the short cycle.

by the same amount as the prematurity of the atrial premature beat and with an identical QRS complex, supporting an antidromic circus movement tachycardia.

The two types of antidromic circus movement tachycardia shown in Figure 2 were sustained. They could be terminated by giving two ventricular premature beats. The second premature beat was not followed by retrograde conduction to the atria, suggesting block in the His-AV nodal pathway as the mechanism of termination.

Figure 3 shows that short episodes of a slower type of antidromic circus movement tachycardia with an RR interval of 440 ms could also be initiated in Patient 1 during right atrial pacing. This tachycardia was characterized by a very long VH interval measuring 280 ms. The tachycardia changed spontaneously into the intermediate and fast rate tachycardias by changes in the VH interval.

Figure 4 illustrates the same phenomenon observed in Patient 2 with a left-sided accessory pathway. The decrease in tachycardia cycle length from 350 to 360 ms to 300 to 280 ms was caused by a shortening of VA time from 210 to 160 ms and resulted in loss of the His potential during the shorter cycles. In all patients ventricular premature beats given during the tachycardia did not change the retrograde atrial activation sequence and were not able to advance atrial activity.

Changes in VH interval during tachycardia (bundle branch reentry). At all rates, the circus movement tachycardias used the accessory bypass as the anterograde limb and the AV node as the retrograde limb of the reentrant circuit. However, they differed in rate because of different subnodal retrograde conduction times. The individual data for all five patients are summarized in Table 1. A possible explanation for this phenomenon can be given by considering different sizes of the reentrant circuit depending on the bundle branch used in retrograde direction during the antidromic circus movement tachycardia. Other observa-

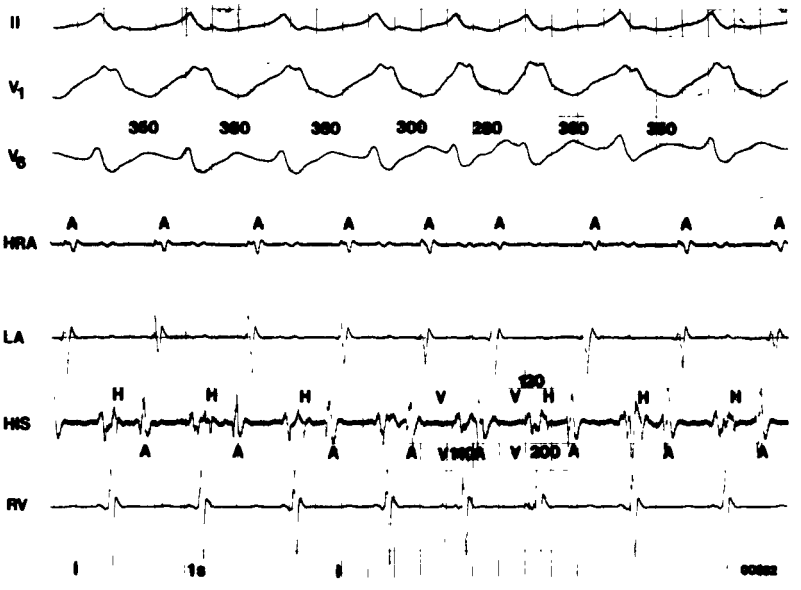


Figure 4. Case 2 (left-sided accessory pathway). Three surface leads (II, V_1, V_6) and four endocavitary leads from the high right atrium (HRA), left atrium (LA), His bundle (HIS) and right ventricle (RV). See text for further explanation.

Table 1. Cycle Length Change in Five Cases of Antidromic Circus Movement Tachycardia

Case	Site of Accessory Pathway	CL (ms)	VA (ms)	VH (ms)
1	Right lateral	440→325→275	330→210→160	280→160→110
2	Left lateral	340→270	230→160	120→?
3	Left lateral	340→290	210→160	130→80*
4	Left lateral	330→280	200→150	140→90*
5	Left lateral	330→160	220→160	160→100

*Unstable His-bundle recording.
CL = cycle length; VA = ventriculoatrial conduction time; VH = ventriculo-His conduction time.

tions made in Patient 1 and illustrated in Figures 5 to 7 show that this mechanism is responsible for the changes in the VH interval during circus movement tachycardia.

During pacing from the right atrium with a basic cycle length of 600 ms (Fig. 5) an atrial premature beat given after 300 ms is conducted in anterograde fashion over the accessory bypass to the ventricle and followed by two QRS complexes. Both nonpaced beats are different in configuration, not only from the beats during atrial stimulation but also from one another. The first QRS complex manifests a left bundle branch block shape and left axis deviation, is preceded by a VH interval of 190 ms and is conducted in retrograde manner to the atrium. The V_2V_3 interval measures 260 ms. The VH interval, preceding the second QRS complex, measures 310 ms and has a V_3V_4 interval of 380 ms. V_4 is also preceded by retrograde activation of the atrium. The configuration of this beat is clearly different from that of the preceding beat, showing a narrower QRS complex and right axis deviation. These observations were reproducible and depended on the achievement of different degrees of VH delay.

We suggest that the mechanism for this phenomenon was bundle branch reentry using different pathways in retrograde direction into the left bundle branch. The atrial premature beat that is conducted over the right-sided accessory pathway to the ventricles and results in a V_1V_2 interval of 300 ms is blocked in a retrograde direction in the right bundle branch. Therefore the impulse travels in retrograde fashion by way of the anterior division of the left bundle branch, resulting in a VH interval of 190 ms. This is followed by reexcitation of the ventricle in anterograde manner over the right bundle branch with a V_2V_3 interval of 260 ms. This ventricular impulse is again conducted retrograde over the left bundle branch system but because of the shorter V_2V_3 interval of 260 ms the impulse is blocked in retrograde manner in the anterior division of the left bundle branch. The impulse is therefore conducted in a retrograde direction exclusively over the posterior division with a VH interval of 310 ms. This allows conduction to the ventricles over the right bundle branch and the anterior division of the left bundle branch and results in a narrower QRS complex with right axis deviation.

This hypothesis was supported by observations made during right ventricular pacing (Fig. 6). During pacing of the right ventricle at a basic cycle length of 770 ms, a ventricular premature beat given after 330 ms (Fig. 6A) was simultaneously conducted in retrograde manner to the atrium over the accessory bypass and to the His bundle over the specific

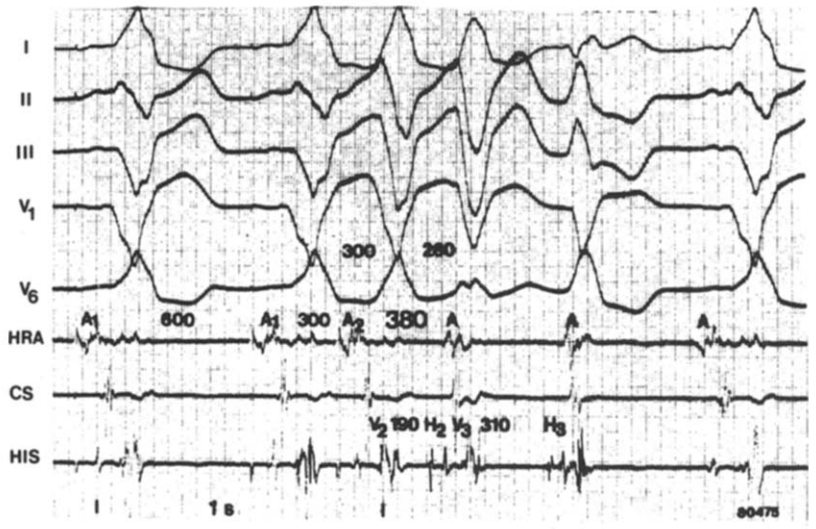


Figure 5. Case 1. During pacing of the right atrium with a basic cycle length of 600 ms an atrial premature beat is given after 300 ms. After conduction to the ventricles this beat is followed by two nonpaced ventricular complexes with a configuration that differs from the beats during atrial pacing and from one another. The first nonpaced ventricular beat with a left bundle branch block shape and left axis deviation appears after a long V_2H_2 interval of 190 ms with a V_2V_3 interval of 260 ms. The second nonpaced ventricular beat has a narrower QRS complex and right axis deviation. It occurs after an increase of the VH interval to 310 ms and with a V_3V_4 interval of 380 ms. See text for explanation of the proposed mechanism of these QRS complexes.

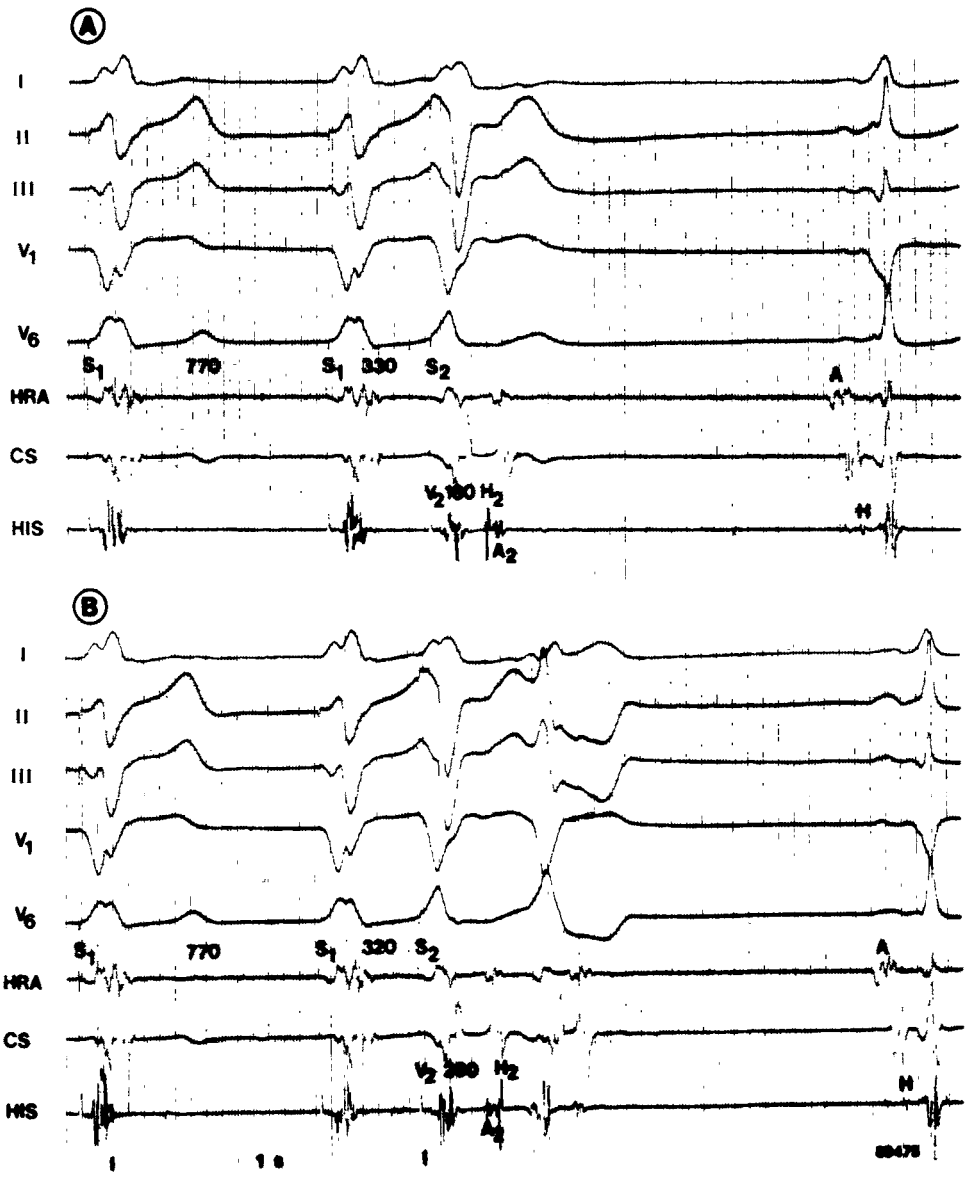


Figure 6. Case 1. Two ventricular premature beat intervals are shown during pacing of the right ventricular apex with a basic cycle length of 770 ms. In **panel A,** a ventricular premature beat given after 330 ms is conducted in retrograde manner to the atria. Atrial activation starts in the high right atrium (HRA), followed almost simultaneously by activation in the His bundle area (HIS) and thereafter in the coronary sinus lead (CS), suggesting activation of the atria over a right-sided accessory pathway. At the same time (**panel B**) activation of the bundle of His occurs via the specific conduction system with a V_2H_2 interval to 260 ms. This is followed by a ventricular beat with a narrower QRS complex and right axis deviation, similar to the beat shown in Figure 3. Note that the retrograde atrial activation sequence of the ventricular premature beat and of the nonpaced ventricular beat is the same as in **panel A,** but that in **panel B** atrial activation occurring after the ventricular premature beat precedes activation of the bundle of His.

conduction system with a VH interval of 180 ms. When the prematurity of the induced ventricular premature beat was increased to 320 ms, a marked increase in subnodal delay was followed by a relatively narrow QRS complex with right axis deviation (Fig. 6B). This QRS complex was similar to the one shown in Figure 6 during right atrial pacing. Note in Figure 6B that activation of the His bundle follows atrial activation, supporting retrograde conduction to the atrium over an AV bypass. We consider it very likely, as shown in Figure 5, that the narrow QRS complex results from bundle branch reentry using exclusively the posterior fascicle of the left bundle branch in a retrograde direction and the right bundle branch and the anterior division of the left bundle branch in an anterograde direction (7). This phenomenon of bundle branch reentry with narrow QRS complex was previously described by Reddy and Khorasanchian (7).

Figure 7 shows that ventricular echo beats that were probably based on bundle branch reentry could easily be initiated during atrial pacing. Panel A illustrates the initiation of two ventricular nonpaced beats occurring after an atrial premature beat during pacing from the high right atrium with a basic cycle length of 770 ms. The atrial premature beat given after 340 ms is conducted anterograde by the accessory pathway and followed by an antidromic echo beat with retrograde conduction over the AV node to the atrium. The VH interval measures 180 ms. This antidromic echo beat is followed by a second nonpaced ventricular beat with a QRS complex with a left bundle branch block configuration and left axis deviation. No delta wave can be recognized at the beginning of this QRS complex. Also, the interval between this QRS complex and the preceding one measures 280 ms and the AA interval is 390 ms. The accompanying increase in the VH interval to 230 ms suggests

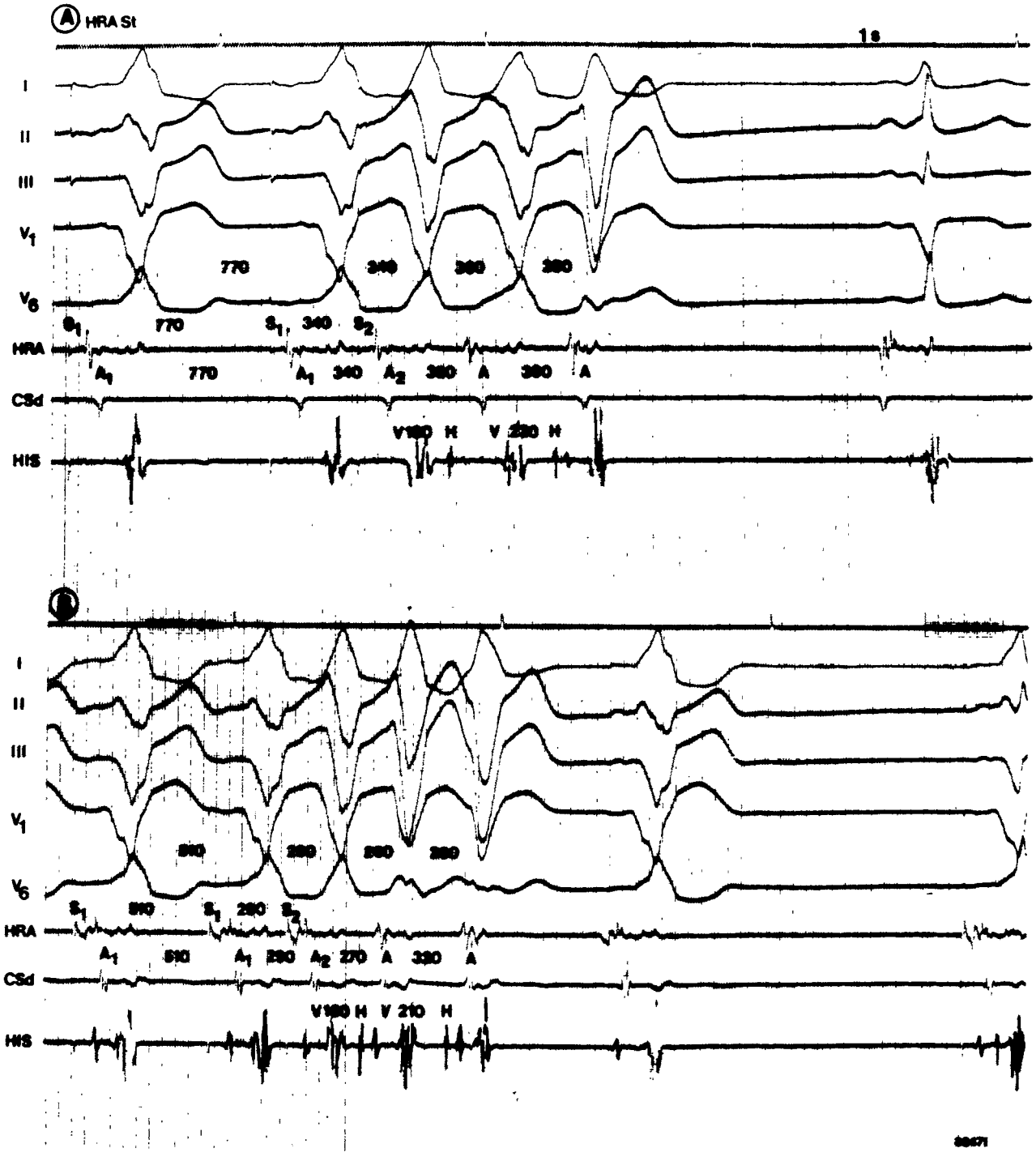


Figure 7. Case 1. **Panel A,** An atrial premature beat (APB) given after 340 ms during basic pacing of the right atrium (HRH St) with a cycle length of 770 ms is followed by two nonpaced ventricular beats. The configuration of the first nonpaced ventricular beat is similar to that of the preceding beats suggesting reactivation of the ventricles via the accessory pathway after retrograde conduction to the atria via the specific conduction system with a VH interval of 180 ms. There is a 1:1 relation between the A_2A and V_2V intervals (both 360 ms). The second nonpaced beat shows a narrower QRS complex, no delta wave and a different shape of the QRS complex. It is preceded by an increase of the VH interval to 230 ms. The VV interval measures 280 ms in comparison with an AA interval of 390 ms. **Panel B,** An atrial premature beat given after 290 ms during basic pacing of the right atrium with a cycle length of 510 ms is followed by two nonpaced ventricular beats with a configuration similar to that of the second ventricular beat in **panel A.** Again, there is no correlation between atrial cycle lengths (A_2A 270 ms and AA 320 ms) and ventricular cycle lengths (VV 260 ms and VV 280 ms) of these beats.

retrograde conduction via the left bundle branch with reexcitation of the ventricle via the right bundle branch. Bundle branch reentry rather than AV junction reentry was considered the underlying mechanism of this QRS complex, because during the stimulation study no evidence was found favoring incremental or decremental conduction properties of the accessory pathway.

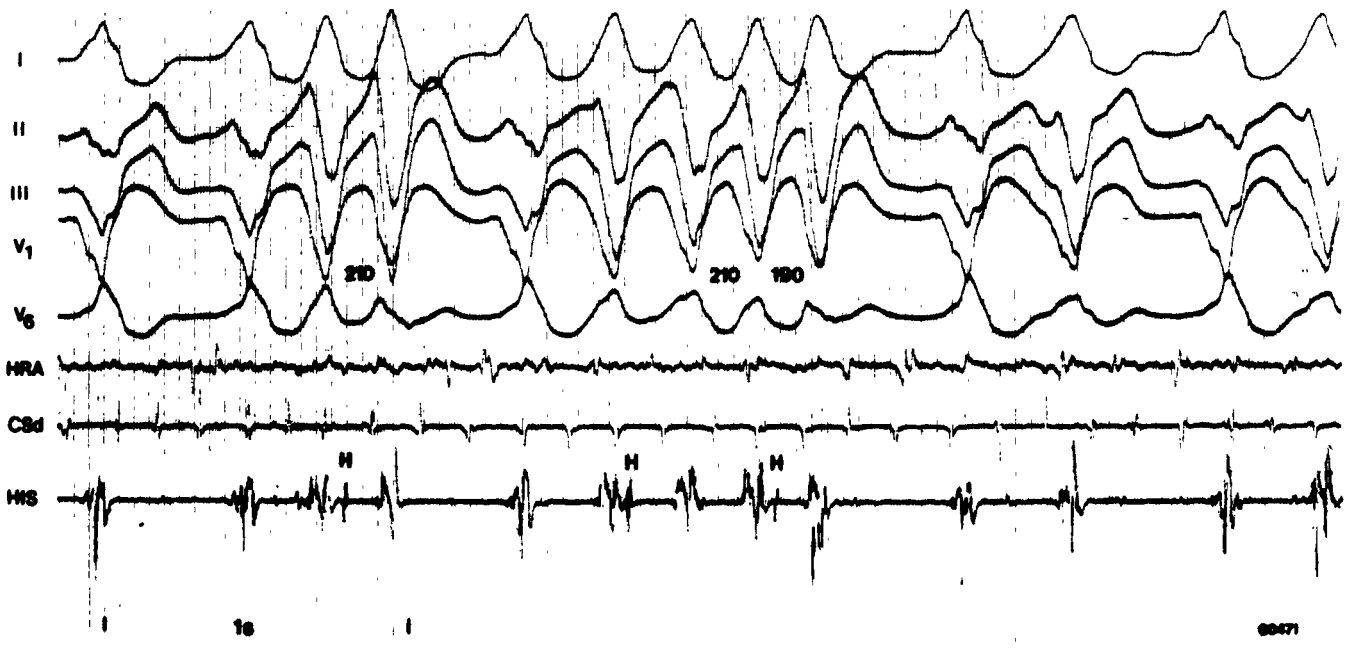
Figure 7B shows similar observations during pacing from the right atrium with a cycle length of 510 ms after administering an atrial premature beat of 290 ms. Again, as observed for the second nonpaced beat in panel A, there is no relation between the atrial and the ventricular cycle length. There is a difference in configuration of the QRS complex of these nonpaced beats in comparison with the preceding beats. This, together with the preceding prolongation in VH intervals, favors bundle branch reentry as the mechanism of the nonpaced QRS complexes.

Observations during atrial fibrillation (Fig. 8). As assessed by the single test stimulus method during atrial pacing with different basic cycle lengths from different sites, the shortest measured anterograde refractory period of the accessory bypass in Patient 1 was 280 ms. However, the shortest RR interval during atrial fibrillation measured 190 ms in this patient. A possible explanation for this difference could be the occurrence of bundle branch reentry during atrial fibrillation. On the surface electrocardiogram during atrial fibrillation the QRS configuration differed after short and long RR intervals. QRS complexes after a short RR interval were narrower as compared with beats ending a long RR interval. The shape of the narrower QRS complexes was very similar to that of the nonpaced ventricular complexes shown in Figure 7 which were thought to be based on bundle branch reentry. Atrial fibrillation was not initiated in the other patients.

Figure 8. Case 1. A recording is shown during atrial fibrillation. The shortest RR interval measures 190 ms. The configuration of the QRS complexes with a long RR interval is different from the configuration of those beats occurring after a short RR interval. The latter complexes resemble the nonpaced ventricular beats shown in Figure 5.

Discussion

Changes in rate of tachycardia based on changes in retrograde conduction. A supraventricular tachycardia with atrioventricular (AV) conduction rather than an accessory AV bypass must be considered in the differentiation of a ventricular and a supraventricular origin of a regular tachycardia with a wide QRS complex. Our five patients had this type of circus movement tachycardia. Sudden changes in the rate of the tachycardia were observed in all. During the electrophysiologic investigation we found that this change in rate was based on changes in ventriculoatrial (VA) conduction time. Because the changes in VH interval occurred suddenly, we postulate the use of different pathways during retrograde subnodal conduction. Because there was no evidence for the presence of additional Kent bundles in these patients, we speculate that the changes in retrograde conduction time were caused by different retrograde entries of the impulse in the bundle branch system. Unfortunately, recordings from the right bundle branch and the fascicles of the left bundle branch were not available to further support this hypothesis.



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It is not understood why retrograde conduction changed from one to another fascicle. Changes in refractoriness after the onset and during the tachycardia, or the fatigue phenomenon in one branch, may have played a role. Another explanation that cannot be definitively ruled out because of lack of bundle branch recordings is the possible presence of Mahaim fibers. One would be very unlikely, however, to find the antidromic type of circus movement tachycardia and Mahaim fibers coincidentally in all five patients with Wolff-Parkinson-White syndrome. We found no evidence for the presence of Mahaim fibers during anterograde AV conduction. Alternatively, changes in conduction velocity using the same pathway should be considered. However, the fact that the VH interval changed suddenly in all patients and in reproducibly defined steps, not only from longer to shorter cycles, but also from shorter to longer cycles (Fig. 4), does not support this explanation.

Above all, the demonstration of different forms of bundle branch reentry in Patient 1 not only during ventricular, but also during atrial pacing seems to us to be the strongest argument to explain differences in conduction time by the use of different parts of the peripheral specific conduction system. All our patients showing this phenomenon had a lateral localization of their accessory pathway. Antidromic circus movement tachycardia was not found in patients with the Wolff-Parkinson-White syndrome with a septally located accessory pathway. Thus, we do not know whether or not the finding of cycle length changes during antidromic tachycardia is characteristic for patients who have lateral accessory pathways.

Determinants of ventricular rate during atrial fibrillation. Factors other than the effective refractory period of the accessory pathway determine the ventricular rate during atrial fibrillation in patients with the Wolff-Parkinson-White syndrome. The duration of this refractory period is, however, of value in identifying patients at risk for life-threatening high ventricular rates when atrial fibrillation supervenes. As previously reported, we (8) and others (2,4) found a good correlation between the duration of the effective refractory period of the accessory bypass and the shortest RR interval during atrial fibrillation. Patient 1 showed a marked discrepancy between the duration of the anterograde effective refractory period of the accessory pathway and the shortest RR interval during atrial fibrillation. Changes in sympathetic tone occurring after the onset of atrial fibrillation may account for shortening of the refractory period of the accessory pathway and an increase in ventricular rate during fibrillation (9).

Our observations suggest the possibility of another mechanism for the occurrence of short RR intervals during atrial fibrillation in patients with the Wolff-Parkinson-White syndrome. After anterograde conduction over the accessory pathway, the impulse may reexcite the ventricle because of bundle branch reentry. This may produce a ventricular rate that is independent of the duration of the anterograde refractory period of the accessory pathway.

Clinical implications. We speculate that the mechanism of cycle length change in antidromic circus movement tachycardia is analogous to slowing of orthodromic circus movement tachycardia during the development of functional bundle branch block ispilateral to a participating accessory AV pathway. In the differential diagnosis of a tachycardia with a wide QRS complex, the finding of a sudden change of the tachycardia rate without change in QRS configuration but accompanied by a shortening or lengthening of the RR interval should immediately suggest the antidromic type of circus movement tachycardia as the underlying mechanism of the tachycardia. We also observed that bundle branch reentry may be a factor determining the ventricular rate during atrial fibrillation in patients with the Wolff-Parkinson-White syndrome. This mechanism could be responsible for a high ventricular rate during atrial fibrillation in patients having a long anterograde refractory period of the accessory pathway.

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